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ABSTRACTS

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AMERICAN REVIEW OF TUBERCULOSIS ABSTRACTS

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Emphysema.—Emphysema is a progressive and incapacitating disease. The majority of cases diagnosed postmortem are not recognized during life. By emphysema is meant a chronic vesicular or hypertrophic condition of the lungs. Acute emphysema, senile emphysema and localized emphysema are probably separate and distinct entities not discussed in this paper. The normal lung lobule or air sac consists of an alveolar duct opening into the atrium, around which are placed the alveoli. In health there is a sudden transition from narrow alveolar duct to wide atrium, so that the inspired air may be thrown as a jet into the more remote parts of the terminal segment. The conclusion is drawn from various observations that the emphysematous lung is deformed in at least three ways. First, many lobules or air sacs are misshapen and the alveolar ducts are dilated, so that air is no longer thrown into the atrium as a jet. Second, the septa between alveoli may disappear so that the supporting framework of the air sac may be lost. Third, the amount of air in the lungs is usually increased, but this increase is not proportionate to the severity of the disease, nor is the volume of the emphysematous lung at rest ever as great as the volume of a normal lung in full inspiration. Thus, whatever may be its microscopical appearance, the lung as a whole in emphysema is not overstretched or overdistended any more than a normal lung is overdistended or overstretched when a breath of moderate depth is taken. The author states that, in his opinion, it is the loss of elasticity of the lungs, coming early and complete that brings about the changes seen in emphysema. The method he used to determine the elasticity of the lung in pulmonary emphysema was the measurement of the intrapleural pressure, which is a direct measure of the tension or elastic recoil of the lung. In emphysema the intrapleural pressure is not always negative as it is in health; even at the end of a full inspiration the intrapleural pressure may be the same as that of the atmosphere, and it is not altered when the lung is collapsed by a pneumothorax. These phenomena can only mean that the elasticity of the lung in emphysema may be entirely lost at a comparatively early stage of the disease. Thus, the loss of pulmonary elasticity in emphysema could be the direct cause of expansion of the thoracic cage, rather than vice versa. Loss of elasticity could also explain the real overdistension of air sacs, with bulla formation which occurs on the surface of the lung. With loss of elasticity the expanding force is no longer equally distributed. In fact, the greatest expansion occurs at the surface of the lung. Thus, with each inspiration the superficial air sacs are stretched and strained to a greater extent than the air sacs deep in the lungs, and, with this process going on for months and years, it is easy to understand the formation of bullae on the surface of the organ. It is agreed that the pulmonary vascular bed is diminished in emphysema and that the work of the right ventricle may be increased, but this does not mean that the total blood flow through the lung is diminished. Microscopically the changes in the lung are uniform throughout the lung, but the dilatation of air sacs is most conspicuous at the periphery. The visceral pleura is thin, flimsy and atrophic.

There is usually a moderate degree of kyphosis, involving all the thoracic vertebrae, and the ribs are widely spaced and run horizontally. By far the most important disturbance of function in emphysema is an inability to ventilate the blood, and this is usually apparent many years before the patient reaches the pathologist. The simplest way to measure deficient ventilation in the lungs is to analyze the arterial blood. The important changes in the blood which occur in emphysema are a gross deficiency in both oxygen absorption and CO_2 elimination. The defect seems to lie in deficient ventilation of the alveoli rather than in any barrier between the alveolar air and the blood. The author rules out the following causes of impaired hemo-respiratory exchange: (*1*) diminution in vital capacity; (*2*) a barrier to the transfer of gases across the pulmonary epithelium; (*3*) insufficient functioning lung due to alveolar destruction; (*4*) diminution in pulmonary blood flow; (*5*) defective ventilation of the pulmonary lobule; (*6*) increase in the volume of lungs; and (*7*) increase in the physiological dead space. The author concludes that the cause of impaired hemo-respiratory exchange in emphysema is the formation of a pathological respiratory dead space, consisting of bullae and other air sacs which are not in contact with the pulmonary circulation. The cause of dyspnea in emphysema is stimulation of the respiratory centre by the CO_2 retained in the blood. In the absence of bronchospasm there is no orthopnea because there is no pulmonary congestion. In attempting to analyze the physical signs in emphysema it is pointed out that the diaphragm, because it is no longer pulled upwards by the lungs, descends and becomes less convex, and its movements on inspiration are greatly diminished. The intercostal muscles which are primarily concerned with moderate inspiration will also be unable to function properly. To expand the chest further the accessory muscles will have to be used, particularly the pectorals, and these raise the front of the chest as a whole in a "heaving" manner. Normally, expiration is largely, if not wholly, a passive act; the thoracic cage is pulled inwards by the elastic recoil of the lung. With loss of elasticity there must be a loss of elastic recoil, so that if the lung is to be deflated it has to be squeezed. Thus, the accessory muscles of the abdominal wall come into play in expiration. The overdistension of superficial air sacs is presumably the cause of the remarkable hyperresonance of the percussion note. Furthermore, with each inspiratory effort the anterior chest wall is pulled outwards and upwards as a whole, and much of this force must be expended on pulling the lung into the pleural sinuses, thus filling the potential gap between the chest wall and the heart. This explains the absence of apical impulse, loss of cardiac dulness, distant heart sounds and loss of liver dulness. The three important factors leading to the formation of the barrel-shaped chest are: (*1*) the inspiratory position of the chest; (*2*) the expansion of the lung between the heart and the sternum; and (*3*) the dorsal kyphosis throughout the thoracic vertebrae. Almost all cases of emphysema have a long-standing history of chronic bronchitis or asthma, or both. The effects of cough and bronchial spasm on the lung and its mechanism are therefore of primary importance in any discussion on the etiology of this disease. During the mechanism of coughing the lung is compressed and not in any sense distended. This building up of pressure followed by its sudden release must mean increased stress and strain on all the structures involved, especially on the alveolar walls, which are not robust and through which passes this wave of pressure change. A chronic bronchitic may cough scores of times a day, and it seems reasonable to suppose that this type of stress and strain repeated over many years could in time produce the loss of elasticity and degenerative changes observed in the alveolar walls. In asthma there is the same degree of change in intraalveolar pressure as in coughing. The author concludes that almost all patients with emphysema suffer from chronic bronchitis or asthma and, thus, the explanation of the pathogenesis of emphysema lies in the effects of those two diseases on the lungs. There still remains to be ex-

plained the unusual case of emphysema with no previous history of asthma or chronic bronchitis. The author can only suggest that, very rarely, the stress and strain on the lung which occurs with ordinary breathing may be sufficient to destroy its elasticity. The author reviewed 72 cases diagnosed on the postmortem table as generalized hypertrophic emphysema. A history of chronic cough was given in 94 per cent and in a quarter of these it was associated with mild or severe asthma. Dyspnea on exertion was present in 78 per cent of the cases. In the majority of the cases, however, the physical signs of emphysema were not observed. In 35 per cent there was enough evidence to suggest an "emphysematous chest," but in only 13 per cent could this evidence be more complete. Thus, the author suggests that a more reliable guide to a diagnosis of emphysema than the physical signs is a history of chronic cough or asthma, associated with dyspnea on exertion. Thus, investigation of a series of 25 patients who gave such a history, and in whom no cause of dyspnea other than emphysema could be discovered, led to the findings that in 92 per cent of them two or more of the physical signs of emphysema were present. In 90 per cent expiration was prolonged, and only 44 per cent showed the traditional picture of emphysema. In the author's opinion the diagnosis of emphysema should only be considered certain when dyspnea on exertion, of insidious onset, not due to bronchospasm or left ventricular failure, appears in a patient who has some of the physical signs of emphysema together with chronic bronchitis or asthma. Treatment of emphysema is essentially symptomatic, as elastic tissue cannot regenerate and nothing can restore the structure of the lungs. Ephedrine not infrequently relieves the dyspnea of emphysema. Respiratory exercises designed to teach the patient to deflate the lung and to increase the use of the diaphragm are important. A well-fitting abdominal belt will also raise the diaphragm by increasing the intraäbdominal pressure. When heart failure supervenes, oxygen should be given. Recovery from heart failure in emphysema is uncommon.—*Emphysema of the Lungs, R. V. Christie, Part I, Brit. M. J., January 22, 1944, 1: 105; Part II, Brit. M. J., January 29, 1944, 1: 143.*—(*D. H. Cohen*)

Emphysema.—Two reasonable hypotheses exist for the development of hypertrophic emphysema: (*1*) the theory of compensatory distension and (*2*) the theory of obstruction. The former reasons that, where alveoli are destroyed by disease, either there ensues a contraction of the thoracic cage or an expansion of the remaining distensible structures or both. The latter theory postulates that, in any disease in which the smaller bronchi and bronchioles are chronically obstructed, as in asthma or chronic bronchitis, there is a difference in the degree of obstruction in the various bronchioles. Therefore, during inspiration, more air will enter the tube of wider calibre than will enter the narrower tube. Hence, alveoli communicating with the less obstructed tubes will inflate more quickly than will the others. In order to fill the less normal alveoli and thus achieve the necessary ventilation, the more normal alveoli will overdistend. This irregularity in distension will produce ultimate loss of elasticity and eventually hypertrophic emphysema.—*The Mechanism of Hypertrophic Pulmonary Emphysema, I. Gordon, Dis. of Chest, May–June, 1944, 10: 180.*—(*K. R. Boucot*)

Emphysema and Lymphoid Hyperplasia.—A case of increasing shortness of breath over a period of three years in a thirty-one year old white male is reported. The patient had been treated for bronchial asthma without success. Physical examination was negative except for hyperresonance with almost complete absence of breath sounds and for marked dyspnea. Roentgenographic examination revealed widely disseminated, healed tuberculosis throughout both upper lobes with some fibrosis and a generalized emphysema. Bronchography and bronchoscopy were negative. The dyspnea and emphysema became increasingly severe and the patient finally died in asphyxia. At autopsy, which was limited to

the chest, emphysema was present throughout the lungs. Masses of lymphoid tissue were present in the walls of small bronchi and bronchioles, frequently bulging into and forming polypoid projections in the lumina, thus producing varying degrees of narrowing. The lungs resembled those of the rabbit or guinea pig. The thymus and mediastinal lymph nodes were normal. The origin and nature of the condition is unknown.—*Bronchiolar Lymphoid Hyperplasia as a Cause of Emphysema: Report of a Case, W. W. Brandes, R. A. Cook & M. P. Osborne, Arch. Path., November, 1943, 36: 465.*—(*D. G. Freiman*)

Bronchial Asthma.—Two cases of bronchial asthma resulting from sulfonamide sensitivity are reported. The first patient developed a status asthmaticus on the tenth day of sulfathiazole therapy for a chronic nasal infection. The patient did not become symptom-free for an entire week. After another week he voluntarily ingested 2 g. of sulfathiazole. After some initial symptoms he developed an attack of bronchial asthma which started four hours after the intake of the drug and lasted for twelve hours. His vital capacity dropped from 4,000 to 2,150 cc. The number of eosinophils showed an initial rise to 13.2 per cent followed by a drop to 4.1 per cent and another rise to 29 per cent after thirty-five hours. This patient had no previous history of an allergic manifestation nor was there a history of an allergic condition in his family. The second patient gave a history of infantile eczema at the age of five years. At the age of thirty years she developed allergic manifestations including asthma due to wheat and house dust. Four years later she took sick with pneumonia and sulfadiazine therapy was given. On the ninth day of chemotherapy a rash was noticed and cyanosis and dyspnea became pronounced. Within twenty-four hours after the discontinuation of the sulfadiazine medication the condition improved markedly. About a year later she received a single dose of 0.5 g. of sulfadiazine for an upper respiratory infection with sore throat. Three hours later she developed a typical asthmatic attack. A trial dose of 0.5 g. of sulfadiazine was voluntarily ingested a year later. There was a drop of her vital capacity from 3,200 to 2,300 cc. but no asthmatic attack developed.—*Bronchial Asthma as a Manifestation of Sulfonamide Sensitivity, T. G. Randolph & F. A. Rawling, J. A. M. A., September 16, 1944, 126: 166.*—(*H. Abeles*)

Silicosis.—A fifty-five year old man offered as chief complaint shortness of breath. Roentgenographic examination of the chest showed enlarged hilar nodes, mottling and accentuated markings. The radiological diagnosis was pneumonoconiosis. The occupational history revealed that the patient had worked as unloader of wheat in a very dusty atmosphere for eight years. The silica content of the dust was 9.96 per cent. Reëxamination of the patient after two years showed progression of the fibrotic process although exposure to the wheat dust had been discontinued.—*A Case of Silicosis Caused by Wheat Dust, T. F. Heatley, D. Kahn & C. R. Rex, J. A. M. A., April 1, 1944, 124: 980.*—(*H. Abeles*)

Bagassosis.—Seven cases of bagassosis, a respiratory illness occurring in people working with bagasse (the product remaining after extraction of sugar from sugar cane), had been reported. Another 11 cases of bagassosis were observed, 2 of which are described in detail. All patients were men; 6 were white, 5 were Negroes. The average age of the patients was twenty-seven years. Exposure to bagasse had taken place from three weeks to two years. Early symptoms were cough and dyspnea. Four patients had hemoptysis. The cough became productive of scant whitish sputum. The patients ran an intermittent fever from 99.8° to 101.2°F., through three or four weeks. Tachycardia was observed in 4 cases. Impaired resonance of the lungs and diminished breath sounds were found in 4 patients, râles in 8. Roentgenological examination revealed miliary mottling throughout both lungs, most dense in the hilar areas. Ten patients developed leucocytosis with 73 to 90 per cent

polymorphonuclear cells. The red cell count and the hemoglobin were usually found increased. The sedimentation rate was increased. Intracutaneous tests with extracts of bagasse were done; allergy could not be proved. Bagasse contains 5 to 7 per cent silica; however, the short incubation period and the short course of the disease speak against silicosis. Evidence from histological study of involved areas of the lung indicates the presence of bagasse dust with a severe and unusual cellular reaction, the nature of which has not yet been established.—*Bagasse Disease of the Lungs, W. A. Sodeman & R. L. Pullen, Arch. Int. Med., May, 1944. 73: 365.*—(*G. C. Leiner*)

Pulmonary Edema.—The incidence of pulmonary edema following exposure to toxic gases has risen due to the increase in industrial production. The most common noxious agents are oxides of nitrogen, phosphorus oxychloride, phosphorus pentachloride, phosphorus trichloride, methyl bromide, chlorine, cadmium and dust from certain alkaloids. A method is described which was used for the prevention and treatment of pulmonary edema. It consists of absolute bed-rest and the immediate administration of oxygen under atmospheric pressure with a provision for expiration against calibrated resistance of from 1 to 6 cm. of water pressure. One starts with 1 cm. of water pressure and increases it to 6 cm. of water pressure within five to ten minutes. Oxygen administration is usually kept up from one to three hours and then it is gradually discontinued. In cases with actual pulmonary edema the recommendation of Richards was followed and a 1 to 100 solution of epinephrine was administered by the oral nebulizer either prior or during the administration of oxygen. The epinephrine can be used continuously with the oxygen or at intervals. This method was used for the treatment of pulmonary edema due to noxious gases in 316 cases during the past year and a half. In some cases the oxygen-helium mixture was used with significant benefit. The routine, however, was to use 100 per cent oxygen to which air could be added. Not more than 25 per cent of air should be added. The hacking cough caused by the oxygen treatment was controlled by one or two swallows of milk. The described form of therapy when used in the incipient stage of pulmonary edema will frequently prevent its advancement to a serious stage. Morphine, venesection or other forms of treatment are of no value or even contraindicated.—*Pulmonary Edema, J. M. Carlisle, J. A. M. A., December 11, 1943, 123: 947.*—(*H. Abeles*)

Spontaneous Pneumothorax in Asthma.—Spontaneous pneumothorax occurred as a complication of bronchial asthma in 2 cases. This is a potentially fatal complication of bronchial asthma. Its diagnosis is extremely difficult in certain cases. This complication should be considered in every case of intractable or atypical asthma. Spontaneous pneumothorax and bronchial asthma are probably sometimes associated with mediastinal and interstitial emphysema. (Author's summary.) —*Spontaneous Pneumothorax Complicating Bronchial Asthma. Report of Two Cases and Consideration of Possible Mechanisms Involved in Its Production, M. Trowbridge, Jr., Arch. Int. Med., June, 1944, 73: 460.*—(*G. C. Leiner*)

Bilateral Spontaneous Pneumothorax.—A twenty-seven year old soldier gave a history of a sudden agonizing pain in the left side of his chest. The previous day he had done some heavy lifting but the pain occurred while resting in bed. Physical and radiological examinations established the diagnosis of spontaneous pneumothorax on the left side. The patient improved on codeine and pentobarbital sodium therapy. Ten days later he experienced a sudden pain in the right side of his chest. He was in acute distress but his condition improved temporarily following decompression on the right and oxygen therapy. Radiological examination revealed marked collapse of the right lung and partial reëxpansion of the left lung. On the eleventh day of hospitalization the patient became

restless, the respiratory rate dropped from 35 to 18 and the respiration was of Cheyne-Stokes character. He died on the same day in deep cyanosis. In the twenty-four-hour period prior to his death he had received a total amount of 0.1 g. of morphine. The post-mortem diagnosis was bilateral pulmonary atelectasis caused by bilateral spontaneous pneumothorax. A small tear was found in the visceral pleura on the left, none could be demonstrated on the right. The heart was displaced to the right, the right ventricle and auricle were much distended. No evidence of tuberculosis was found.—*Bilateral Spontaneous Pneumothorax, A. C. King & M. Benson, J. A. M. A., July 15, 1944, 125: 782.*—(*H. Abeles*)

Spontaneous Pneumothorax.—The 33 cases of spontaneous pneumothorax observed were thus subdivided: spontaneous pneumothorax probably due to tuberculosis, 24.2 per cent; spontaneous pneumothorax definitely due to tuberculosis, 33.3 per cent; spontaneous pneumothorax due to bronchial asthma and empysema, 11.1 per cent; spontaneous pneumothorax due to other pulmonary conditions, 18.1 per cent. The remaining 12.1 per cent were observed in young persons without pulmonary discax and without previous history.—*Considerazioni critiche su 33 casi di pnx. spontaneo, Marfori & Austonu, Il Policlinico, 1939, 46: 113.*—(*G. Simmons*)

Spontaneous Pneumothorax.—In a brief survey of the literature Itard is credited with first use of the term "pneumothorax" in 1803. His and subsequent work indicated that it occurred spontaneously mainly in tuberculous patients. But in 1887 Hall suggested its occurrence in nontuberculous individuals and in 1942 Ornstein and Lercher reported 58 cases in apparently healthy persons. Chief theories as to its causation are: (*1*) rupture of a pleural tuberculous focus, (*2*) rupture of a pleural adhesion, (*3*) rupture of an emphysematous bleb and (*4*) presence of a pleural defect. In 50,000 examinations of the chest reviewed by Santos and Tanchonco an incidence of 0.11 per cent was noted, males predominating two to one. Ehrlich and Schomer classified spontaneous pneumothorax thus: (*1*) the open type with air freely entering and leaving the pleural cavity through a rupture of the lung, (*2*) the closed type with only a transient admission of air into the pleural cavity before sealing of the rupture and (*3*) the valvular type with air entering the pleural space during inspiration and being trapped there to cause a tension pneumothorax. McGuire and Bean described a fourth in which air escaped from ruptured alveoli or bronchi into pulmonary interstitial tissue. Cardinal symptoms are chest pain and dyspnea but shock and cyanosis may be present in tension pneumothorax. Diagnosis may be made on physical examination alone but radiographic confirmation is advisable. A period of bed-rest with subsequent avoidance of physical efforts, especially exertion with a closed glottis, is given as the treatment of choice; aspiration of air should be resorted to only in the presence of definite tension. X-ray examination to rule out underlying pulmonary disease is advised before discharge of the patient. The authors report 5 cases of spontaneous pneumothorax, all in apparently healthy members of military units. A tuberculous focus was not found in any one of the 5; so it was assumed that a ruptured emphysematous bleb was the etiological agent in all. Four gave a history of exertion at the time of collapse while the fifth was at complete rest. All were recommended for reclassification as regards military service and it is suggested that every person who undergoes spontaneous pneumothorax be so reclassified. —*Spontaneous Pneumothorax, G. H. Stein, E. B. McConkie & A. J. Kuhn, War Med., September, 1943, 4: 324.*—(*L. R. Roll*)

Bronchial Carcinoma.—Carcinoma of the lung ranks next to carcinoma of the colon in frequency in men. The first signs or symptoms of bronchial carcinoma consist of any change from normality, and the commonest of these is cough. At first the cough is usually dry and unproductive and is dismissed

as "smoker's cough," "bronchial catarrh" or some such vague condition. By far the most important group of symptoms arises from the infection that sooner or later follows upon bronchial obstruction. It often begins as an apparently straight-forward attack of pneumonia or "influenza" but significantly resolves incompletely or not at all. "Delayed resolution," "unresolved pneumonia," recurrence of smouldering sepsis in part of a lung, particularly in a patient of middle age, should always give rise to a strong suspicion of malignant lung disease. General deterioration of health, loss of weight and loss of strength are usually the signs of approaching death and, so far as operability is concerned, their presence almost always means that the case is unsuitable. Once the presence of a growth of the lung is suspected, good chest radiograms should be taken. Any suspicious shadow revealed will demand further investigation by bronchoscopy. During bronchoscopy a piece of tissue should be removed for biopsy. The only cure for bronchial carcinoma is by operation. Operability at present is rather less than 10 per cent. The operation of choice is pneumonectomy. The recurrence rate after only lobectomy is unduly high. The operative mortality in this series was 29 per cent, but included were 3 cases over sixty years of age. Pneumonectomy performed properly for carcinoma in a patient under sixty should not carry an operative risk over 10 per cent, however. Of the 29 cases in this series, 8 died from operation, 7 from recurrences and 14 are alive and well. The disability from a successful pneumonectomy is negligible. Of the 21 cases surviving operation, all but one had sound healing and only one needed a thoracoplasty. All of the successful cases returned to their former occupation which in some cases was quite arduous. The main complaint is occasional dyspnea on overexertion.—*Surgical Treatment of Bronchial Carcinoma, B. C. Brock, Brit. M. J., August 28, 1943, 2: 257.*—(*D. H. Cohen*)

Bronchial Origin of "Alveolar Cell Tumor."—There is a small group of primary carcinomata of the lung described under a variety of names, but always with the idea that the primary foci are the septal cells lining the pulmonary alveoli. Unfortunately there is no agreement as to just what kind of cells line the pulmonary alveoli. Current views range all the way from a continuous layer of epithelial cells; an epithelial but discontinuous layer; that the cells are not epithelial but mesenchymal in origin; and that there are both epithelial and mesenchymal cells present. One of the most conspicuous and consistent findings in any chronic inflammatory or fibrotic process of the lung is the presence of cuboidal epithelium forming a continuous lining of the alveolar septa or spaces in a fibrous tissue stroma. The histogenesis of the epithelium has two interpretations. The older and by far the more prevalent view is that the cells arise from former alveolar epithelium which has now resumed its embryonic appearance. The second view is that these cells do not originate from the septal cells but that they are outgrowths of the basal cells of the bronchiolar mucous membrane. In an effort to establish the histogenesis of these regenerated cells, a detailed review was made of 60 bronchiectatic lungs together with miscellaneous cases of tuberculosis, organizing pneumonia, interstitial pneumonia and lipoid pneumonia. The two most important and constantly observed changes relevant to the present discussion were (*1*) the frequent occurrence of metaplasia of the bronchial mucosa to a transitional or squamous cell type and (*2*) the presence of various amounts of granulation or fibrous tissue which consistently surrounded the involved smaller bronchi and their terminal ramifications, and the gradual merging of this tissue with the adjoining thickened alveolar septa. Throughout these areas, regular, low cuboidal or even attenuated epithelium lined the alveoli and the spaces within the granulation and fibrous tissue. On consecutive sectioning these lining cells were seen to originate in the mucosa of the bronchioles. Furthermore, since preoperatively lipoidol was instilled in all of the

lungs, there were frequently seen large macrophages whose cytoplasm was distended with injected oil completely filling the alveoli lined by regenerated cuboidal cells. If the regenerated alveolar epithelium arises from septal cells, one would also expect these regenerated cells to show at least some degree of phagocytosis, since septal cells are known to be phagocytic. But none of the latter showed any evidence of phagocytosis. All available evidence substantiates the supposition that the source of the regenerated alveolar epithelium is the basal cell of the bronchioli and not the preëxisting cell which has now resumed its embryonic appearance. The microscopic findings from 6 cases of "alveolar cell tumor" of the lung are discussed in detail. All of these cases apparently showed many or most of the alveoli lined with cuboidal or columnar cells. Often the tumor cells lined only a portion and one side of an alveolus and then ended abruptly. *Conclusions:* Considerable evidence is presented to show that regenerated alveolar epithelium arises not from septal cells but from the basal cells of the bronchioles. On this basis alveolar cell tumors are also considered to arise from the basal cells of the bronchioles and not from septal cells. This is given additional support from a study of the pleomorphism of the more commonly observed bronchogenic carcinoma in which squamous or anaplastic cells and cuboidal or columnar cells are found side by side in the same tumor and even in the same microscopic field. It is believed that the parent cell in all cases of primary carcinoma of the lung is the basal cell of the bronchial or bronchiolar mucosa. The distribution of the subsequent tumor is dependent upon the further differentiation of the cells. If they are anaplastic or squamous, they will be either irregularly distributed throughout the lungs or will occupy the alveolar spaces just as does an inflammatory exudate in pneumonia. If they are cuboidal or columnar, they will regularly line the septa producing the well known alveolar arrangement. While only a few tumors are indisputably of one group or the other, there are many intermediary transitions between the two. (With 3 plates.).—*Bronchiolar Origin of "Alveolar Cell Tumor" of the Lung, P. A. Herbut, Am. J. Path., September, 1944, 20: 911.*—(*J. S. Woolley*)

New Growths of Chest.—Neoplasms of the chest may be classified as follows: (*1*) tumors of the major bronchi, (*2*) tumors of the minor bronchi or periphery of lung, (*3*) anterior mediastinal tumors, (*4*) posterior mediastinal tumors, (*5*) rare mediastinal tumors, (*6*) tumors originating in the chest wall, (*7*) non-neoplastic tumors. Subdivisions under these main headings are presented. As diagnostic criteria, diagnosis by biopsy and cell study is recommended. Material may be obtained by endoscopy, lymph node removal, punch biopsy of the tumor proper, centrifuged material from pleural fluid or sputum. X-ray studies with flat plates, stereoscopy and fluoroscopy are recommended. Bronchography may be helpful. Tomography may demonstrate the relationship between the tumor and the bronchus. Diagnostic pneumothorax may differentiate pulmonary from mediastinal or thoracic wall tumors. Diagnostic pneumoperitoneum may distinguish between supra- and subdiaphragmatic lesions, including herniation. Deep X-ray treatment may help to differentiate the lymphoblastomata. X-ray studies of possible primary tumors may confirm suspicion of metastatic malignancies. Bronchoscopy and esophagoscopy may be diagnostic. Surgical exploration is warranted. Laboratory tests to rule out non-neoplastic diseases such as tuberculosis, leukemia, thyroid enlargement, etc. is important when indicated. Kymography and thoracoscopy may assist in the diagnosis of pulsating masses and pleural tumors, respectively. Symptoms and signs are of considerable significance. Thirteen case studies are presented.—*New Growths of the Chest, C. W. Tempel, Dis. of Chest, July-August, 1944, 10: 277.*—(*K. R. Boucot*)

Congenital Absence of Lung.—Authors here report 2 cases, one proved by exploratory

operation, and 38 other cases from the literature. There may be three types of this abnormality: (*1*) true aplasia in which there is no trace of lung, bronchus or vascular supply; (*2*) the bronchus is represented by a small outpocketing from the trachea but no lung tissue is present; (*3*) there is extreme hypoplasia, the bronchus is fully formed but is reduced in size and ends in a fleshy structure without lobes which lies within the mediastinum. The symptoms may be only dyspnea, cyanosis and harsh breathing; young patients usually do not develop so well or so rapidly as normal children do. Objectively there are found: asymmetry of the chest, scoliosis (either toward or away from the affected side), atrophy of muscles, displacement of heart and mediastinum toward the side of the absent lung, elevation of the diaphragm and narrowing of intercostal spaces. The percussion note may be dull or flat, or resonant from hypertrophy and emphysema of the other lung. Breath sounds are either absent or suppressed or bronchial for the same reason. Bronchoscopic studies reveal a block of the main stem bronchus a short distance from the carina provided there is a rudimentary main stem bronchus at all. Absence of a lung is not incompatible with life nor does it preclude a long life. In the literature 25 patients were children under twelve years of age. Eleven lived to be more than nineteen years old, and 3 patients reported were fifty-eight, sixty-five, and seventy-two years of age. This anomaly is more common on the left side.—*Agenesis of the Lung, A. R. Valle & E. A. Graham, J. Thoracic Surg., August, 1944, 13: 345.*—(*W. M. G. Jones*)

Resection of Vagus Nerve.—A case of a thirty-five year old soldier is reported in whom widening of the posterior superior mediastinum to the left of the midline was found on a routine chest film. The diagnosis of neurofibroma was suggested by the location of the tumor and by the physical examination which revealed numerous *café au lait* spots. The patient was asymptomatic. On operation, a pedunculated mass, 10 cm. in diameter, originating from the sympathetic nerve trunk was found in the posterior superior sulcus. There were two more tumors, 5 cm. in diameter, both originating from the left vagus nerve. All three tumors and a 15 cm. long portion of the left vagus nerve were removed. Histological section of the operative specimen confirmed the diagnosis of neurofibroma. The postoperative course was uneventful with the exception of a transient Horner's syndrome and paralysis of the left vocal cord.—*Resection of the Left Vagus Nerve for Multiple Intrathoracic Neurofibromas, B. Blades & D. J. Dugan, J. A. M. A., October 16, 1943, 123: 409.*—(*H. Abeles*)

Eosinophilic Pleuritis.—The true inflammatory nature of eosinophilic infiltrations has been amply proved on the basis of autopsy material. Pleural effusions accompanying these infiltrations are rare but some have been reported. Usually the effusion is not large enough to permit examination of a specimen. In a twenty-one year old soldier with a typical eosinophilic infiltrate of both lungs, bilateral pleural effusions developed. Diagnostic tap was performed, and in each instance amber clear fluid was obtained. On the right side the fluid contained 850 leucocytes with 29 per cent eosinophils. On the left side there were 2,600 white cells with 64 per cent eosinophils. The fluid was sterile on culture, including guinea pig examination for tubercle bacilli. Otherwise the disease ran a typical course with blood eosinophilia, eosinophils in the sputum, and with complete recovery. Thirteen weeks after onset of the illness many ova of ascaris were found in the stools, and in the following week two adult worms were passed following a vermifuge. After this the residual blood eosinophilia disappeared completely, although the cutaneous reaction for ascaris allergy remained strong. In the above case the causal connection between the ascaris infestation and the eosinophilic lung infiltrations is clear. Ascaris infestation is a common cause of this disease in Switzerland but undoubtedly other allergens are capable of

producing an identical pathological picture.—*Eosinophile Pleuritis bei flüchtigem, eosinophilen Lungeninfiltrat, H. Baumann, Schweiz. med. Wchnschr., April 1, 1944, 74: 326.*—(*H. Marcus*)

Chylothorax.—Many cases of chylothorax are not recognized. It should be suspected whenever a milky fluid is obtained at thoracocentesis, especially if there is no obvious reason for the presence of pleural fluid. This milkiness is due to presence of excess fat and is pathognomonic of chylothorax. The fat may be recognized by the fact that it tends to rise as a creamy layer on standing; the fat stains readily by Sudan III; if the fluid is alkalinized and shaken with ether the fluid clears; and chemical examination will show a fat content of 0.4 to 4.0 per cent. The lower values will be found in cases of hydrothorax which may occur in association with chylothorax. The usual bacteriologic and pathologic studies of pleural fluid are not of assistance in diagnosis. Chylous ascites and rarely chylopericardium may also occur in association with chylothorax. The etiology of chylothorax is varied: trauma, severe coughing, gunshot wounds, blast injuries, and overvigorous attempts to resuscitate newborn infants. It may occur following thoracic duct injury in the course of surgical operations. In nontraumatic cases malignant disease is often responsible or tuberculosis. Filariasis may cause it. In the cases secondary to trauma there is usually a delay in onset, two to ten days, probably caused by its collection first in an extrapleural pocket, until rupture of the pleura takes place. Then dyspnea and shock are prominent symptoms. The accumulation of chyle may be massive and will require repeated thoracocentesis. Dramatic relief is often experienced by this measure. Loss of large amounts of fluid produce dehydration and emaciation and even death in a few days. Development of peripheral edema in chronic cases is due to loss of serum proteins. In about 50 per cent of cases, healing of the ruptured duct takes place in several weeks. Spontaneous rupture of the duct with similar features and mortality in children has also been reported. In nontraumatic cases, the clinical features are similar but it is insidious in onset. The prognosis is generally poor, especially in the nontraumatic forms, most frequently due to malignant disease. There is no satisfactory method of treatment. A high carbohydrate, high protein diet is advised. Aspirated chyle (using sodium citrate or heparin as anticoagulants) has been given intravenously but an occasional death has resulted. A safer method of restoring the blood osmotic pressure is to use blood plasma. Surgical closure of the ruptured ducts has not been successful. The introduction of sterile broth or gomenol into the pleural space to aid in the closure by production of an aseptic pleuritis has been reported. Diaphragmatic elevation by phrenic crushing cured one reported case. X-ray therapy of underlying malignant disease has benefited some cases.—*Chylothorax, A. M. Olsen & G. T. Wilson, J. Thoracic Surg., February, 1944, 13: 53.*—(*W. M. G. Jones*)

Acute Empyema.—The treatment of pleural empyema is simple drainage; the drainage must remain adequate for as long as there is an abscess cavity. The time at which an empyema should be drained is a matter of clinical judgment. Aspiration should be performed frequently, slowly and without air replacement. When an empyema is ready for draining a liberal incision should be made, at least two inches of rib removed subperiosteally and a tube inserted. Pleural empyema has a bad reputation for healing, merely because of the lack of equal elasticity of the cavity wall. Here the resultant varying changes are due to movements of the intercostal muscles, the diaphragm and the chest wall. The author lists the following series of causes of inadequate drainage: (*1*) obstruction by fibrinous masses within the drainage tube; (*2*) obstruction by granulation tissue; (*3*) obstruction by kinking of tube; (*4*) tube too short to reach the pleura; (*5*) tube through the chest wall but not reaching the cavity;

(*6*) tube too far into cavity; (*7*) part of the cavity below the drainage opening; (*8*) loculation. When an empyema shows evidence of unsatisfactory progress the patient should be set upright in bed and the wound inspected. The tube should be removed. If a free flow of pus results then one realizes that the tube was too long. Never use force in probing a sinus because of the danger of air embolus, temporary paralysis or cerebral abscess. An uncomplicated acute empyema, kept properly drained and properly dressed, should pursue an uninterrupted course to recovery. If such steady progress is interrupted by a febrile attack, one is too often prone to use the sulphonamides and not investigate the tube and wound. The most likely cause of fever is inadequate drainage. The author stresses the importance of early physiotherapy to prevent contractures of the chest wall and interference with respiratory function. Breathing exercises and exercises for the major thoracic muscles are used. The author notes the large incidence of infected hemothorax due to improper treatment of the original condition. He states that blood and bloody fluid in the chest which result from trauma should be aspirated without air replacement. When infection is once established the chest should be opened, all clot evacuated and adequate drainage established. —*Management of Acute Pleural Empyema, P. R. Allison, Brit. M. J., September 25, 1943, 2: 383.*—(*D. H. Cohen*)

Chronic Empyema.—The records of 346 patients with chronic empyema at the Mayo Clinic were studied to learn the cause of the chronicity and the methods used to eradicate it. Chronic empyema is not a disease but a complication of pneumonia, influenza, pleurisy, tuberculosis, trauma and acute exanthemata, in the order named. The most common causes of chronicity are (*1*) inadequate drainage, (*2*) tuberculosis, (*3*) too late drainage (that is, empyema not recognized early), (*4*) bronchial fistula and (*5*) bronchiectasis. There was no demonstrable correlation between clinical type of antecedent illness and type of bacteria cultured from empyema cavity. It is safe to perform open drainage for pneumococcic pus if the specific gravity is 1,040. In streptococcic empyema the underlying lung infection must be combated first. Closed drainage or aspirations must be used until the specific gravity of the pus is 1,035–1,040. While most cases showed pleural thickening in X-ray films, in 6 per cent there was no visible pleural reaction. Prophylaxis of chronic empyema is the establishment of early, adequate drainage in every case of acute empyema. To be adequate, drainage must involve the removal of a portion of at least one rib. The more radical the drainage the shorter the patient's stay in the hospital and the lower the mortality rate. In cases with a thoracic sinus, pleural biopsy should be done to discover tuberculous infection, and a more radical drainage employed. The presence of bronchial fistula does not increase the mortality or morbidity rates, and many of them will heal spontaneously or with minor surgical attention. Closed drainage cured only 8 per cent but is of greater value as it keeps a cavity drained until the mediastinum becomes fixed and open drainage becomes safe. Seventy-one per cent of these cases had open drainage. Thoracoplasty was employed for only 5 per cent, as this is a very mutilating operation and should never be used unless absolutely necessary. The size of the cavity should be measured frequently (by filling the space with saline solution) and X-ray films taken also to record the progressive decrease in size of empyema. Irrigation of the space with Dakin's solution twice daily is recommended as an aid in lung expansion. If conservative treatment does not do this, more radical procedures (unroofing cavity or thoracoplasty) should be considered. In cases of mixed tuberculous empyema, thoracoplasty is particularly valuable and should be used early as lung fibrosis prevents lung reëxpansion. Muscle transplants were used to fill spaces impossible to collapse by thoracoplasty (2 cases). A draining sinus may remain after almost total obliteration of the empyema cavity. Spontaneous healing of

sinus may occur but should be complete in a year. The causes of persistence of sinus are continuance of the chronic empyema, bronchocutaneous fistula, tuberculosis, the presence of a foreign body, sclerosis of the walls of a sinus and osteomyelitis of a contiguous rib. In this series, 46 patients (out of the 212) had a draining sinus, but some of these were of short duration and may be healed by now. There were 18 deaths in the hospital and 3 deaths at home of causes related to the empyema. These deaths were from operative shock and spread of the infection by contiguity or metastasis, chiefly brain abscess. Of the 212 patients who answered a written inquiry, 164 stated their health was excellent and that they had resumed their usual mode of life.—*Chronic Empyema, O. T. Chagett & V. D. Shepard, J. Thoracic Surg., June, 1943, 12: 464.*—(*W. M. G. Jones*)

Unusual Cause of Chronic Empyema.—The author lists the causes of chronic empyema as: inadequate drainage, foreign body, bronchopleural fistula, inexpandable lung, tuberculosis and fungus infections. He emphasizes another cause—dermoid tumors of the mediastinum, and presents 2 cases. The pleural involvement follows secondary infection within the lung. In both cases, tube drainage had been previously carried out for the empyema, and there were residual broncho-pleuro-cutaneous fistulae. Resection of the dermoid tumors resulted in cure in each case. Careful diagnostic study is stressed. Tubercle bacilli and fungi are sought in the sputum and sinus discharges by culture and guinea pig inoculation. X-ray films are taken in postero-anterior and lateral views with lipiodol injection of the sinuses. Dermoids are usually found in the anterior mediastinum, while the posterior costovertebral gutter is usually free of involvement. Fluoroscopy of stomach and colon are also suggested to rule out diaphragmatic hernia. Material from the sinus is examined for hair, etc. Because of malignant changes in at least 15 per cent of these tumors, early surgical removal is advised.—*Chronic Empyema Due to Dermoid Tumors of the Mediastinum, J. M. Dorsey, Surgery, May, 1943, 13: 755.*—(*D. J. Rednor*)

Treatment of Empyema.—The main obstacle to the successful treatment of empyema by the closed method is the presence on the pleura of a thick layer of fibrinous, purulent exudate. Sodium tetradecyl sulfate is capable of liquefying this exudate, thereby paving the way for bacteriostatic agents. Among the latter, sodium sulfadiazine proved satisfactory, when used together with azochloramide which oxidizes the sulfonamide inhibiting peptones and para-aminobenzoic acid. The combination of azochloramide and a sulfonamide (sulfanilamide) had a definite bacteriostatic effect on *S. aureus in vitro. In vivo* the author uses 80 grains (5.2 g.) of sodium sulfadiazine in 100 to 200 cc. of azochloramide-tetradecyl solution, and leaves this mixture in the pleural cavity till the next irrigation. Physiological saline is employed as irrigating fluid. Sulfadiazine blood levels should be determined and the blood picture and the urine examined at frequent intervals. So far, no toxic signs were encountered. The case history of a thirty-six year old white female is given. Empyema (staphylococcal) developed in the course of pneumothorax treatment. After saline irrigations and penicillin therapy had failed to bring about improvement, azochloramide-tetradecyl-sulfadiazine treatment was instituted and continued weekly for three and one-half months. After five weeks the pleural fluid became clear and cultures turned negative. No relapse occurred up to date. Six other cases are under similar treatment.—*A Suggested Therapeutic Procedure for the Treatment of Empyema by the Closed Method: A Preliminary Report, E. E. Carpenter, Journal-Lancet, August, 1944, 64: 278.*—(*P. Lowy*)

Clotted Hemothorax.—Clotted hemothorax is a frequent complication of chest trauma. In a review of 426 cases of hemothorax, 10 per cent were clotted. The pathogenesis of the condition is not known. Infection is

said to favor clotting, but fully half of the operated cases were uninfected. Two types of hemothorax are seen. The more common clotted hemothorax is a multiloculated fibrino-hemothorax. The massive clot is less common. Physical signs are those of fluid or of consolidation in the latter type. The diagnosis depends on difficult or unsuccessful aspiration in a case which obviously has fluid in the pleural space. Small basal clots which produce no fever are left alone. Indications for thoracotomy are large clots, persistent temperature elevation, multiloculated fibrino-hemothorax and clotted hemothorax with foreign body. When the entire lung is collapsed, no time is lost in operating. When the upper lobe is expanded, the formation of adhesions to the upper lobe is awaited before thoracotomy. The optimum time is somewhere between three and five weeks following injury, but cases from two weeks to two months have been operated on. After a time interval longer than two months reëxpansion of the lung cannot be expected. The pleural space is approached through an intercostal incision. A section of the sixth rib is sometimes resected for better exposure. The clot is evacuated and the dense fibrin coat overlying the visceral pleura is removed. If this is not done lung expansion is slow or incomplete. The fibrin layer is incised, and it can usually be lifted away from the visceral pleura by finding the plane of cleavage with the finger. Oozing from the visceral pleura is rarely severe. It is severe, however, from the parietal pleura, and the fibrin there is not removed other than from the costphrenic sinus and part of the diaphragm. Sodium penicillin is instilled following the operation, and the drain which is placed into the wound is not opened until twelve hours postoperatively. In uninfected cases it can usually be removed on the fifth day, when the discharge is less than one ounce. The results are best in uninfected cases, and there were no fatalities in this group. Even the empyema group does remarkably well, and only one case died of *Staphylococcus aureus* septicemia several weeks postoperatively.—*Clotted Hemothorax, R. W. Lush & J. C. Nicholson, Lancet, October 7, 1944, 247: 467.*—(*H. Marcus*)

Intrathoracic Foreign Bodies.—At a Forward General Hospital, 30 per cent of all severe chest casualties had retained foreign bodies. Out of 92 cases seen with foreign bodies, 50 were operated on, and the foreign body was removed in 49 instances. Removal of the foreign body is always advised if it is more than one cm. in diameter, or if it is of a jagged appearance. There were 2 deaths following the operation, one in which the missile was embedded in the heart and could not be recovered following pericardiotomy, and one in which the missile was in the right upper lobe where it had caused abscess formation and septic erosion of the ascending aorta. The best time for operation is the earliest moment at which the patient's condition permits. Ten days after wounding is the average, unless the foreign body is pleural and an obvious empyema is present. Then the foreign body is removed at the time the empyema is drained. Accurate X-ray localization is of prime importance. Intrapulmonary or mediastinal foreign bodies are best demonstrated by anteroposterior and lateral films. If the missile is near the chest wall, fluoroscopic examination is necessary to decide whether the missile is intrathoracic or extrathoracic. Thoracotomy with rib resection provides the usual approach. The exact site depends on the location of the foreign body. For small intrapleural or superficial intrapulmonary foreign bodies, intercostal incision may suffice. When there is no gross evidence of infection the chest is closed without drainage. Penicillin is used in infected cases, and in cases who develop infected effusions later on. Such effusions are handled by needle aspirations. Thorough removal of blood clot and fibrin from the pleural space at the time of operation is indicated.—*Intrathoracic Metallic Foreign Bodies, A. L. D'Abreu, J. W. Litchfield & C. J. Hodson, Lancet, August 26, 1944, 247: 265.*—(*H. Marcus*)

Wounds of Chest.—Chest injuries are divided, for clinical purposes, into nonpene-

trating wounds and penetrating wounds. The nonpenetrating wounds include contusions and open wounds which do not penetrate the parietal pleura. The superficial open wounds should not be treated by complete excision since the relationship of the soft tissue layers may be markedly different at the time of treatment from the one at the time the injury was sustained. Penetrating wounds when of minimal extent and without complications are best treated by immobilization with adhesive strapping and morphine to assure rest. The patient is kept in bed in Fowler's position for several days. Large wounds of the thoracic wall may produce a serious picture caused by paradoxical respiration. In addition, the mediastinal flutter may cause twisting of the mediastinal structures inducing circulatory embarrassment and reflexes on the vasomotor system resulting in shock. Treatment consists in immediate closure of the wound by anything at hand followed by the usual measures to combat shock. After the shock has been overcome attention is being paid to further treatment of the chest wound. Under field block anesthesia or, if necessary, under inhalation anesthesia, preferably with cyclopropane, the wound is excised, bleeding controlled, the visceral pleura closely inspected for tears, sulfanilamide instilled into the pleural cavity and the wound is closed with stay sutures. If there was obvious contamination of the pleural cavity closed drainage may be established. Following closure of the wound the lung is expanded by aspiration of air and by using positive pressure. Another serious injury results when a valve-like mechanism is produced by the wound. Marked respiratory embarrassment is caused by the resulting tension pneumothorax. Circulatory distress is less prominent in this type of chest injury. Treatment consists of under-water decompression by needle or catheter; the latter is preferable if there is a simultaneous hemothorax or lung injury. If the chest injury is complicated by an internal hemorrhage, usually from an intercostal vessel or from an internal mammary vessel, conservative treatment is to be preferred. If respiratory embarrassment is produced the hemothorax should be gradually decompressed with simultaneous air replacement in order to keep up the pressure tamponade. Not more than 500 cc. of blood should be removed at one time. Many serious chest injuries are complicated by injuries of the lung. Tension pneumothorax or hemopneumothorax are the most common complications. When the pulmonary damage produces shock which does not respond to the usual therapeutic measures, radical procedures have to be employed since it was demonstrated by operative and autopsy findings that there is usually gross interstitial pulmonary hemorrhage present. Another indication for surgical intervention is damage to the great pulmonary vessels. The nature of the surgical procedure may vary from ligation of a vessel to pneumonectomy depending upon the findings in the individual case. If a communication of the abdominal cavity with the pleural cavity is produced by the injury the abdomen should be explored first and then a thoracotomy with catheter drainage should be performed. Injuries to the intrapericardial structures may produce cardiac tamponade manifested by the Beck triad, a falling arterial pressure, a rising venous pressure and a quiet heart; paradoxical pulse, distention of veins of the neck and reduction of cardiac excursions on fluoroscopic examination may also be present. Cardiac tamponade will not develop when the blood can enter the pleural or abdominal cavity through the opening in the pericardium but severe hemorrhagic shock will dominate the clinical picture. In these cases the clinician may suspect a cardiac injury by the position of the wound of entrance and the profound degree of shock. Treatment consists in closure of the heart wound and evacuation of the pericardial contents. The death rate from wounds of the heart is about 23 per cent.—*Wounds of the Chest, E. J. McGrath, J. A. M. A., February 19, 1944, 124: 488.*—(*H. Abeles*)

Thoracic Injuries.—Thomas describes the nature and treatment of thoracic injuries in the forward area of war zones where operative facilities are not the elaborate ones found in relatively stationary areas. The more serious thoracic injuries often prove fatal before there is any possibility of removal to an operating unit. The cause of death of these cases, apart from shock and hemorrhage, is severe interference with the cardio-respiratory mechanism which continues to operate as a cause of death during the first forty-eight hours after receipt of the injury. If the patient survives this period, the cause of death is generally a direct result of superimposed infection. Shock is a variable factor in chest injuries and is usually in proportion to the degree of tissue damage. In many cases the simple anti-shock remedies of warmth, both externally and internally, of hot drinks with morphia to control pain will be enough. Oxygen therapy is a valuable measure, particularly if there is any respiratory embarrassment. The absence of shock is no indication for allowing the patient freedom of movement. The chief function of the forward unit is to relieve immediate symptoms caused by cardio-respiratory embarrassment and secondarily to institute treatment which will decrease the incidence of complications. The commonest causes of interference with normal cardio-respiratory function are: (*1*) open pneumothorax, (*2*) tension pneumothorax and (*3*) much more rarely tamponade of the heart by blood in the pericardium. These three conditions will demand adequate treatment. Surgical emphysema, a common occurrence, is usually of limited extent and has little clinical importance. The severe type is most often the result of an underlying tension pneumothorax. Stove-in chest may constitute a serious threat to the patient from the paradoxical movement of the chest wall. It may result from crush injuries and is commonly associated with a hemothorax and acute dilatation of the stomach. The commonest condition met which does not menace life in the early course is hemo- or hemopneumothorax with or without a retained foreign body. Under ideal conditions, the object of treatment should be the removal of every medium of infection, that is, foreign bodies of all kinds, removal of all damaged tissue and rapid re-expansion of the lung. In a forward area, however, this is not possible so that a certain percentage of cases develop late infection which require later more extended treatment in a thoracic centre. Conservative treatment of hemothorax involves aspiration without gas replacement. The author gives pertinent reasons why gas replacement is contraindicated. Concerning the use of sulfonamides, the author deprecates the tendency to depend too much on chemotherapeutic aids at the expense of violating basic surgical principles. —*Thoracic Injuries: The Rôle of the General Surgeon in the Forward Area, C. P. Thomas, Brit. J. Tuberc., July–October, 1943, 37: 103.*—(*E. H. Rubin*)

Chest Wounds.—The author records his experience at a casualty clearing station in the immediate treatment of thoracic casualties. The aim was to ensure that the patient had recovered sufficiently to withstand the rigors of evacuation to a chest centre. The material consists of 127 consecutive battle casualties with penetrating wounds of the chest. The majority had passed through a surgical unit, but treatment had been confined to dressing and the administration of morphine. Those admitted within forty-eight hours of wounding comprised 46 per cent of the cases; those within seventy-two hours 72 per cent. An analysis of the intrathoracic status revealed that 88 (69.4 per cent) had haemothorax and/or hemopneumothorax—5 of these were fatal; 13 (10.2 per cent) had thoraco-abdominal injuries—6 of these died; 14 (11.0 per cent) had pulmonary hematoma; 5 (3.9 per cent) each fell in the groups of pneumothorax and atelectasis and there were 2 cases of lung abscess. In hemothorax the bleeding is seldom of long duration and is checked by pulmonary collapse. The presence of blood in the pleural cavity provokes a

serous reaction. In case of hemopneumothorax the air may enter the pleural space from within or from without. Only one case of tension pneumothorax was noted. Infection was the earliest complication occurring in 9 of the 88 cases. In each case the infection was caused by the *Str. hemolyticus*, and was established within seven days of wounding. The ideal treatment in traumatic hemothorax is to empty the pleural space—with or without air replacement depending on symptoms. Repeated aspirations were done in the cases of infective hemothorax, but in 5 cases closed drainage was necessary. The patients received 4 g. of sulphanilamide daily for six days. Of these 88 cases, 5 were fatal before evacuation; none died subsequently. Atelectasis was noted in 5 cases and was probably a sequel of bronchial obstruction by aspirated blood or secretion. Treatment of these 5 cases was confined to attempts to moderate the pain and to promote drainage by posture; infection was discouraged by sulphapyridine. In all 5 cases uneventful re-aeration was achieved. The author's experience leads him to conclude that the immediate treatment of the parietal wound should be as conservative as possible. The indications are to control hemorrhage from the chest wall and to close an open pneumothorax. If there is no serious hemorrhage and the wound is not "sucking," simple dressing is adequate; if the wound is "sucking" it should be closed by strapping over an air-tight pad and the intrapleural pressure lowered by aspiration of the hemothorax.—*The Early Treatment of Wounds of the Chest in the Middle East, R. B. Scott, Brit. M. J., April 8, 1944, 1: 490.*—(*D. H. Cohen*)

Closure of Open Chest Wounds.—A muscle plastic operation is described for the closure of the wide open chest following the Schede operation. The accepted treatment for tuberculous empyema is radical surgery: thoracotomy, thoracoplasty and Schede operations. The anatomical result is a wide open chest with exposed lung, taking months and years to close if spontaneous closure takes place at all. A muscle plastic closure cannot be satisfactorily accomplished at the time of the Schede operation. The reasons are: the toxemic condition of the patient with an empyema cavity and the failure of muscle flaps to adhere in the presence of severe infection, especially if the tubercle bacillus is a causative factor. The operation should be planned far in advance so that at the time of the thoracoplasty and Schede procedures, a minimum amount of extracostal musculature will be traumatized or sacrificed. Small residual empyema cavities may be closed by resecting the scapula and implanting a pedicled flap of scapular and latissimus dorsi muscles. Larger chest defects require, in addition, a graft of erector spinae muscle to cover the exposed lung. Postoperative wounds heal by primary union as early as four to six weeks. On the other hand, if left to close spontaneously, patients with open chest wounds remain in the hospital as long as three years. Six patients have been operated upon according to this technique, with success in 5 cases and failure in one. All 5 patients are well enough to earn a livelihood in part at least, and one patient went through childbirth without incident. The unsuccessful result occurred in an emaciated individual, weighing about 75 pounds, who had an unrecognized caseous focus in his lung at the time of operation. The result was extensive tuberculous infection in the wound and a hematogenous dissemination throughout the body. The principles involved in this operation have been successfully used by the author in treating patients with chest defects resulting from nontuberculous empyema. The absence of tuberculous inflammation in such cases makes for less chance of complications and more rapid wound healing. (Author's Summary.)—*Closure of Open Chest following the Schede Operation for Tuberculous Empyema, M. Weinstein, Surg., Gynec. & Obst., August, 1944, 79: 148.*—(*G. C. Leiner*)

Penetrating Chest Wounds.—In a general hospital, located midway between the forward base hospital and the chest centre in England—

the mortality from chest wounds and their complications is relatively low. Patients whose condition is good enough to be removed from the forward hospital to a general hospital have a good chance to survive. In a series of 260 cases, 15 deaths were recorded. Empyema was by far the most common serious complication; it was the contributing cause of death in 13 of the fatal cases. Hemothorax was the most frequent mild complication; it was present 125 times. It is treated by prompt aspiration which is repeated if necessary. Small amounts of air are injected only if the patient's comfort demands it or if the X-ray localization of pockets is to be facilitated. If clotting of the blood occurs in the chest, the clots are usually left undisturbed. Complete reëxpansion of the lung is the rule. However, clotting in a hemothorax frequently foreshadows infection which has to be treated vigorously. Pyothorax was the most common serious complication. It was present 77 times. It is treated by repeated needle aspiration and instillation of sodium penicillin. If the infecting organisms are gram positive cocci or clostridia, the results of penicillin treatment are very good. In loculated empyemata needle aspirations are insufficient and rib resection and "nettoyage" are done. Adhesions between pockets are broken at operation and large pieces of fibrin are removed from the pleural cavity. Intrapleural and intrapulmonary foreign bodies are removed at the same time. After operation, underwater drainage through a tube is provided. With adequate drainage, lung expansion is the rule, even in cases with bronchopleural fistula. Drainage must be continued not only until discharge ceases, but until it can be demonstrated by the injection of contrast medium that the empyema cavity has become obliterated. Atelectasis of one or more lobes occurred 44 times. Reëxpansion is the usual outcome of this complication. Extrapleural hemothorax was found 15 times and was treated by aspiration. All foreign bodies, whether intrapleural or intrapulmonary, were removed if they were over 1 cm. in diameter, provided that the patient's condition permitted. Thoraco-abdominal wounds were present 34 times. Two of these patients died. Treatment of these wounds must include suture of hollow viscera through an abdominal, a thoracic or a combined approach. Subphrenic abscess was a complication 7 times, and all patients recovered following drainage of the abscess. When the diaphragm is injured it is sutured at the time the empyema is drained. Penicillin has also proved of great value in infected wounds of the chest wall where it is used locally in the form of calcium penicillin. It can be administered through Carrel-Dakin tubes which have been sewed into place. When pyopneumothorax is associated with infected chest wounds penicillin is also given parenterally. In the treatment of empyema, however, parenteral administration is useless because the thick pleura acts as a barrier.—*Major Complications of Penetrating Wounds of the Chest, J. W. Litchfield & C. J. Hodson, Lancet, August 12, 1944, 247: 197.*—(*H. Marcus*)

Pectus Excavatum.—The author presents the only 2 entirely successful cases operated on at the Massachusetts General Hospital. The operation was according to the principles put forth by Dr. A. Lincoln Brown. The patients were two sisters, fourteen and five, the elder suffering from dyspnea, the younger symptomless. In both, the deformity was steadily becoming more prominent. The operation consists primarily of removing appreciable lengths of the lower costal cartilages and a wedge of bone at the upper limits of the gladiolus. A vertical midline incision is used, the xiphoid process removed and the rectus muscles reflected from their attachments. The diaphragmatic attachments are then cut and the costal cartilages divided up to the second or third rib. A transverse wedge of bone is then removed from the sternum just below the articulations of the second or third costal cartilages. Wire sutures are used to hold the trimmed lower portion of the sternum up in the proper position. Large segments of costal cartilage are

then removed and held in place by heavy silk sutures. In closing the skin, the redundant portion is removed. At the end of the operation a light breast plate of plaster was moulded to fit the anterior chest wall to prevent paradoxical motion. No external traction was employed.—*Pectus Excavatum: Report of Two Cases Successfully Operated On, R. H. Sweet, Ann. Surg., June, 1944, 119: 922.*—(*D. J. Rednor*)

Anesthesia in Bronchoscopy.—To obviate the unpleasantness of the bronchoscopic procedure for the patient, intravenous pentothal anesthesia has been advocated. The patient is given premedication of morphine and scopolamine or atropine. Eight cc. of a 5 per cent pentothal solution are injected over a thirty-second period. To avoid the danger of laryngeal spasm and cessation of respiration, 10 per cent carbon dioxide and oxygen are given through a tube, and the anesthetic is injected only when the patient's respiration becomes hyperpneic. The larynx is exposed with the laryngoscope as soon as the patient's jaw muscles relax, and when the glottis is open the bronchoscope is introduced immediately. Otherwise this event is awaited. During the procedure, 10 per cent carbon dioxide in oxygen may be administered continuously through a tube hooked into the corner of the mouth. This gas mixture is administered again through the side tube of the bronchoscope just before the instrument is withdrawn through the larynx. With this procedure, withdrawal spasm is usually avoided, but if necessary the gas mixture can be given through a positive pressure mask after the bronchoscopy. The advantages of the procedure are mainly those of the comfort of the patient, especially in case of repeated bronchoscopic treatments. The disadvantages and contraindications are those of pentothal anesthesia in general. It is inadvisable in cases presenting respiratory difficulties, an additional cocainization may be necessary in patients who present technical difficulties due to configuration of mouth, jaw and neck.—*Pentothal Anaesthesia in Bronchoscopy, L. Fatti & H. J. V. Morton, Lancet, May 6, 1944, 246: 597.*—(*H. Marcus*)

Spontaneous Diaphragmatic Paralysis.—Spontaneous paralysis of the phrenic nerve, particularly in children, is not a very common occurrence. In the case reported, X-ray films revealed paralysis of the left half of the diaphragm in a nine year old child. In addition, there were enlarged hilar lymph nodes on the left and a thickened interlobar fissure, suggestive of a residual interlobar effusion. It is thought that the paralysis was caused by compression either by the lymph nodes, the pleural effusion or both. Anatomical conditions are such that a paralysis of the phrenic nerve may occur more readily on the right than on the left. The commonly expressed opinion that spontaneous paralysis may be caused by enlarged lymph nodes alone, however, does not satisfy the author, because in that case the vagus too should be involved by the compression. Paralysis of the vagus, however, is an extremely rare condition. The author concludes that in the case reported there was an abnormality in the anatomical course of the left phrenic nerve which made it possible for the enlarged lymph nodes to compress the nerve.—*Su un caso di paralisi spontanea dell'emidiaframma sinistro in un bambino, A. Traversa, Riv. di tisiol., 1940, 13: 206.*—(*G. Simmons*)

Pneumopericardium.—A forty-two-day-old Negro infant was readmitted to the hospital thirty-two days after birth because of abscess of the right buttock. The baby had had a healthy father, but her young mother had had a positive Wassermann during pregnancy and had undergone antiluetic therapy consisting of a series of bismuth and neoarsphenamine injections. Delivery was difficult due to the large size of the infant. White asphyxia was present, requiring artificial respiration and the use of respiratory stimulants. Vitamin K and 20 cc. of blood had been given into the right buttock. The immediate postnatal hospital course was eleven days due to some transitory neurological abnormalities. The

abscess of the right buttock was incised after readmission, but it failed to heal. Instead it progressed to rectal fistula formation. There then occurred an episode of acute dyspnea accompanied by bubbling râles and distant heart sounds. The febrile course was unaltered by sulfapyridine therapy, so a chest X-ray film was taken. This revealed pneumopericardium, disseminated bronchopneumonia and two large abscesses in the right lower lobe. Following a second attack of dyspnea, the child died. Autopsy confirmed the X-ray findings, gas escaping under tension when the parietal pericardium was incised. The larger of the two abscesses was in intimate contact with the pericardium and was also in communication with a fairly large bronchus. Staphylococci were recovered from the lung abscess.—*Pneumopericardium in a Forty-two Day Old Infant, Am. J. Dis. Child., April, 1944, 67: 288.*—(*K. R. Boucot*)

Dilatation of Pulmonary Artery.—Congenital dilatation of the pulmonary artery is rare. Recognition of this condition rarely occurs before necropsy. Radiology plays an important part in the diagnosis of this condition, and the criteria of X-ray diagnosis seem to be as follows: (*1*) increase of pulmonary bow; (*2*) small aortic knuckle; (*3*) broadening of hilar shadows; (*4*) enlargement of the pulmonary arterial tree. The authors give a clinical history of a patient believed to have the above named condition. He is a man of twenty-three who was admitted for influenza, but routine examination revealed certain peculiar features. He was 6 ft. 5½ in. in height and had clubbed fingers and a blue tinge on his lips. He had been told in childhood that he had "heart trouble." He had no illnesses and lost no time from schooling. Three years before admission he had a bout of blood spitting which was not repeated. He gives no history of rheumatic fever. Examination of the heart reveals the apex beat to be well out. There was a palpable diastolic thrill over the precordium. On auscultation there was a mid-diastolic murmur at the apex. The lungs were clear. Blood pressure 140/70; RBC 7.5 million; Hb over 140 per cent (Sahli). The electrocardiogram revealed diaphasic QRS complexes. X-ray examination demonstrated a huge pulmonary artery with a comma-shaped branch, also greatly enlarged in the antero-posterior position. After a barium swallow a very large impression made by the right pulmonary artery in the right oblique position was a striking feature.—*A Case of Congenital Dilatation of the Pulmonary Artery, H. L. Heimann & M. M. Posel, Brit. M. J., October 23, 1943, 2: 512.*—(*D. H. Cohen*)

Lupus Erythematodes Familialis.—Familial lupus erythematodes is very rare. Twenty-one cases of lupus erythematodes occurring in 2 or 3 members of a family have been reported; 17 times the occurrence in brothers and sisters has been seen, only 4 times in parents and children. This report concerns 3 sisters, forty-five, forty-eight and fifty-four years of age, with the same localizations (nose, jaws) and with the same clinical form of the disease (discoid). Improvement took place in all 3 of them after almost the same treatment.—*Lupus Erythematodes Familialis, A. D. Jaworowska, Urol. & Cutan. Rev., July, 1944, 48: 333.*—(*G. C. Leiner*)

Chronic Myeloid Leukemia.—A case is reported of chronic myeloid leukemia which was diagnosed in November, 1935, and was followed in Lakeside Hospital Out-Patient Department between eight periods of hospitalization. The patient was treated intermittently with Fowler's solution and on three occasions received roentgen ray therapy. There was a good response to both types of treatment. Five months prior to death pleurisy developed on the left following a head cold, and X-ray examination revealed a pleural effusion. Serial X-ray films revealed an increase in the pleural effusion and, subsequently, parenchymal infiltration. The pleural fluid was positive for tubercle bacilli. During the final four weeks of illness the patient received nine to twelve minims of Fowler's solution daily. No X-ray therapy

had been given during the preceding eight months. On the day of death the white blood count was 12,850 per cc.; the differential count, 82 per cent polynuclears, 9 per cent myelocytes, 2 per cent myeloblasts, 5 per cent basophils, one per cent lymphocytes and one per cent monocytes. Autopsy revealed miliary tuberculosis of the lungs, liver, spleen, kidneys, bone marrow, lymph nodes and adrenal glands; active primary tuberculosis of upper left lung and hilar lymph nodes; myeloid metaplasia of bone marrow; splenomegaly, hepatomegaly and chronic adhesive fibrous pleurisy. Bone marrow sections from sternum, vertebra and long bones showed only myeloid hyperplasia and metaplasia. There were increased numbers of myeloid elements without a marked increase in immature forms, and all the marrow contained adequate numbers of red blood elements and megacaryocytes. Leukemic infiltration in other organs was completely absent even in the spleen. It is impossible to say whether these findings were due to the drug or the infection, but it is felt that the infection probably played an important part and may have produced some factor during the course of the leukemia causing the reticulo-endothelial system to revert towards normal. If the mechanism of this process were known, the pathogenesis of leukemia might be more readily discernible.—*Morphologic Obliteration of Chronic Myeloid Leukemia by Active Tuberculosis, R. W. Heinle & D. R. Weir, Am. J. M. Sc., April, 1944, 207: 450.*—(*G. F. Mitchell*)

Regional Enteritis.—This disease of unknown etiology is recognized as a clinical and pathological entity by characteristic gross and microscopic changes of the involved bowel. The lesion starts as an acute inflammation in the intestinal mucosa, proceeds to ulceration and, in the chronic stages, to stenosis and fistula formation. Patchiness of involvment is characteristic. Healthy bowel alternates with severely disease segments, and it is common to find lesions of various ages in one segment, so that all phases of the disease may at times be observed in one specimen. When the mucosa is involved, it presents shallow ulcers with a vascular granulation tissue as their base. The villi are often hypertrophic. The submucosa is edematous and infiltrated with a cell-rich exudate. The muscularis mucosae shows relatively few changes. A neuromuscular hyperplasia of its component elements is commonly seen, and the same reaction is encountered in the outer muscular coats. It is unclear whether the hyperplasia of the nervous elements of Auerbach's plexus is only apparent due to contraction of involved segments or whether it is real. The serosa shows striking changes in and around the lymphatic channels, amounting frequently to occlusion of the latter. Miliary serosal nodules with epithelioid cells and giant cells are fairly characteristic, and these often lead to a mistaken diagnosis of tuberculous enteritis. Tubercle bacilli have never been demonstrated in these lesions. Cellular infiltration along the lymphatic channels of the mesentery and hyperplasia of the nodes draining the affected bowel are common. The involvment of the lymphatic systems suggests that obstruction of the lymphatic drainage may play a rôle in the etiology of the disease.—*Regional Enteritis, F. Owens, Arch. Surg., June, 1944, 48: 472.*—(*H. Marcus*)